DISTURBANCES IN NUCLEOPROTEIN METABOLISM

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Relevance: Nucleoproteins are fundamental structural units of the cell nucleus, consisting of nucleic acids associated with specific proteins. They play a critical role in the storage and transmission of genetic information, as well as the overarching regulation of cellular metabolism. Pathological disturbances in nucleoprotein turnover lead to the dysregulation of purine degradation, resulting in hyperuricemia (elevated serum uric acid). This metabolic imbalance causes the systemic deposition of urate salts, triggering significant structural and functional damage. Clinically, this is most commonly observed in gout, renal complications, and urolithiasis.

Objective: To identify and analyze the morphological and structural alterations in cells and tissues resulting from impaired nucleoprotein metabolism. The study aims to investigate the underlying pathogenetic mechanisms and clarify their role in metabolic diseases to enhance early diagnosis and evidence-based clinical management.

Materials and Methods: The breakdown of nucleoproteins releases purine bases, which are ultimately metabolized into uric acid. Excessive production or impaired renal excretion of uric acid leads to the precipitation of sodium urate crystals within various tissues.

Musculoskeletal System: In joint tissues, urate accumulation leads to gout. This process is characterized by the formation of tophi—granulomatous nodules with a crystalline urate core surrounded by inflammatory cells (macrophages and giant cells) and fibrous tissue. Over time, this results in the destruction of articular cartilage and subchondral bone.

Renal System: Urate salts primarily deposit within the renal tubules and the interstitium. This causes tubular obstruction, interstitial inflammation, and progressive fibrosis, which gradually compromises renal function.

Hepatic Impact: Chronic hyperuricemia can induce "micro-tophi" in the liver, increasing cellular oxidative stress and disrupting lipid metabolism and detoxification pathways. These changes are vital markers in the pathogenesis of systemic conditions like urate infarction.

Conclusion: Impaired nucleoprotein metabolism, driven by disrupted purine pathways, results in hyperuricemia and the systemic deposition of urate crystals. These events lead to irreversible morphological changes and the clinical manifestation of diseases such as gout and urolithiasis. Understanding these pathways is essential for developing effective protocols for the early detection and treatment of metabolic disorders.